5. POTENTIAL FOR HUMAN EXPOSURE

5.1 OVERVIEW

α-, β-, γ-, and δ-HCH have been found in at least 112, 128, 163, and 109 of the 1,467 current or former EPA National Priorities List (NPL) hazardous waste sites, respectively (HazDat 1998). The frequency of these sites within the United States can be seen in Figures 5-1, 5-2, 5-3, and 5-4.

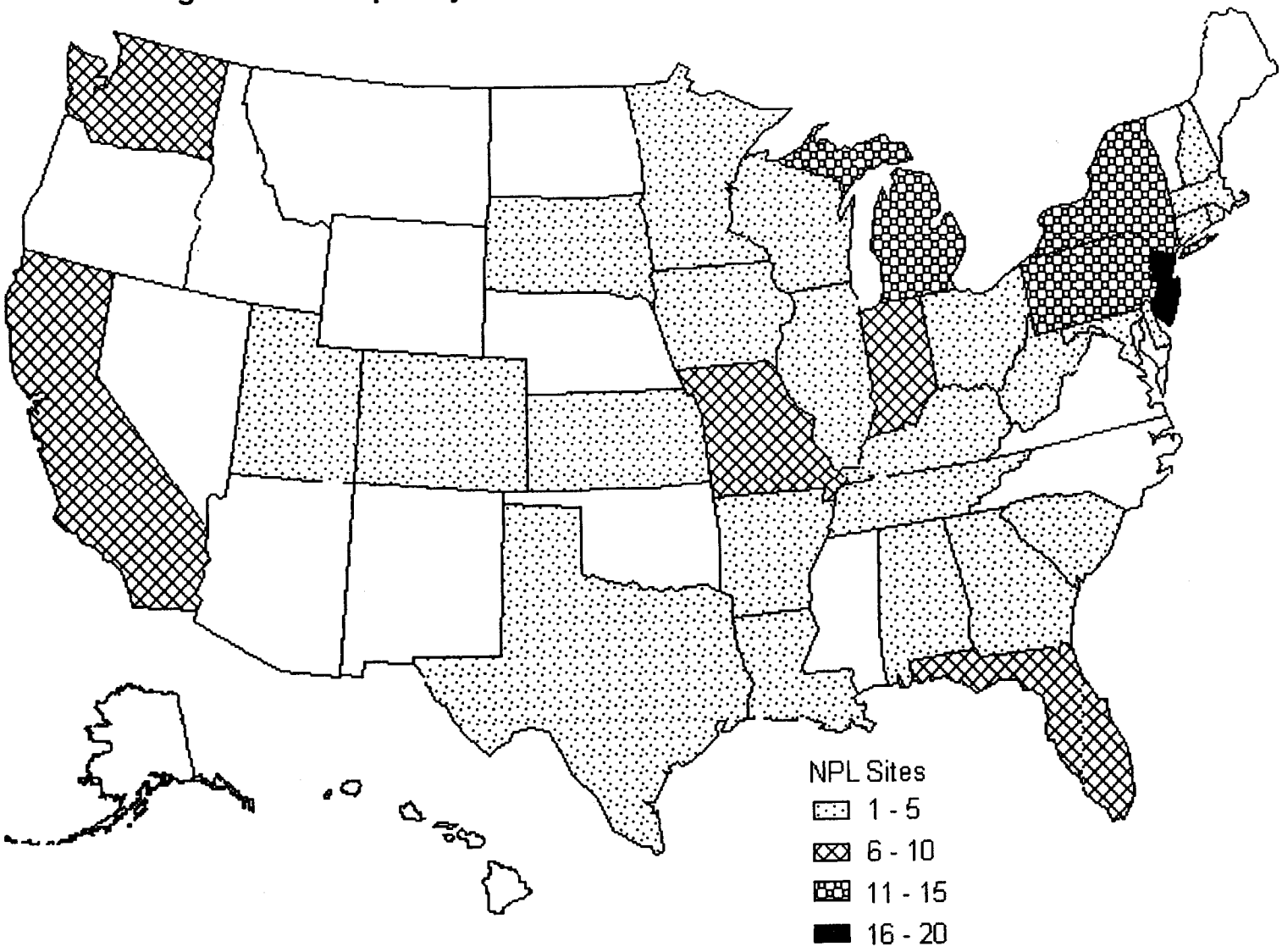
HCH can be released to the environment during the formulation process and through its use as a pesticide. Once released to the environment, HCH can partition to all environmental media. HCH in the atmosphere, either as a vapor or adsorbed to particulates, can be photolytically degraded but is primarily removed from the atmosphere by rain-out and dry deposition. Biodegradation is believed to be the dominant decomposition process for HCH in soil and water. The rates of degradation depend on the ambient environmental conditions. HCH has been detected in air, surface water, groundwater, sediment, soil, fish and other aquatic organisms, wildlife, food, and humans. Human exposure results primarily from medicinal use and from ingestion of contaminated plants, animals, and animal products. HCH has not been found to be a major contaminant of drinking water supplies.

5.2 RELEASES TO THE ENVIRONMENT

According to the Toxic Chemical Release Inventory, in 1996, releases of lindane to the environment from four large processing facilities were 2,429 kg (5,397 pounds) (TRI96 1998). In addition, an estimated 1,664 kg (3,697 pounds) were transferred offsite (TRI96 1998). There were no releases to publicly owned treatment works (TRI96 1998). Table 5-1 lists amounts released from these facilities. The TRI data should be used with caution because only certain types of facilities are required to report. This is not an exhaustive list.

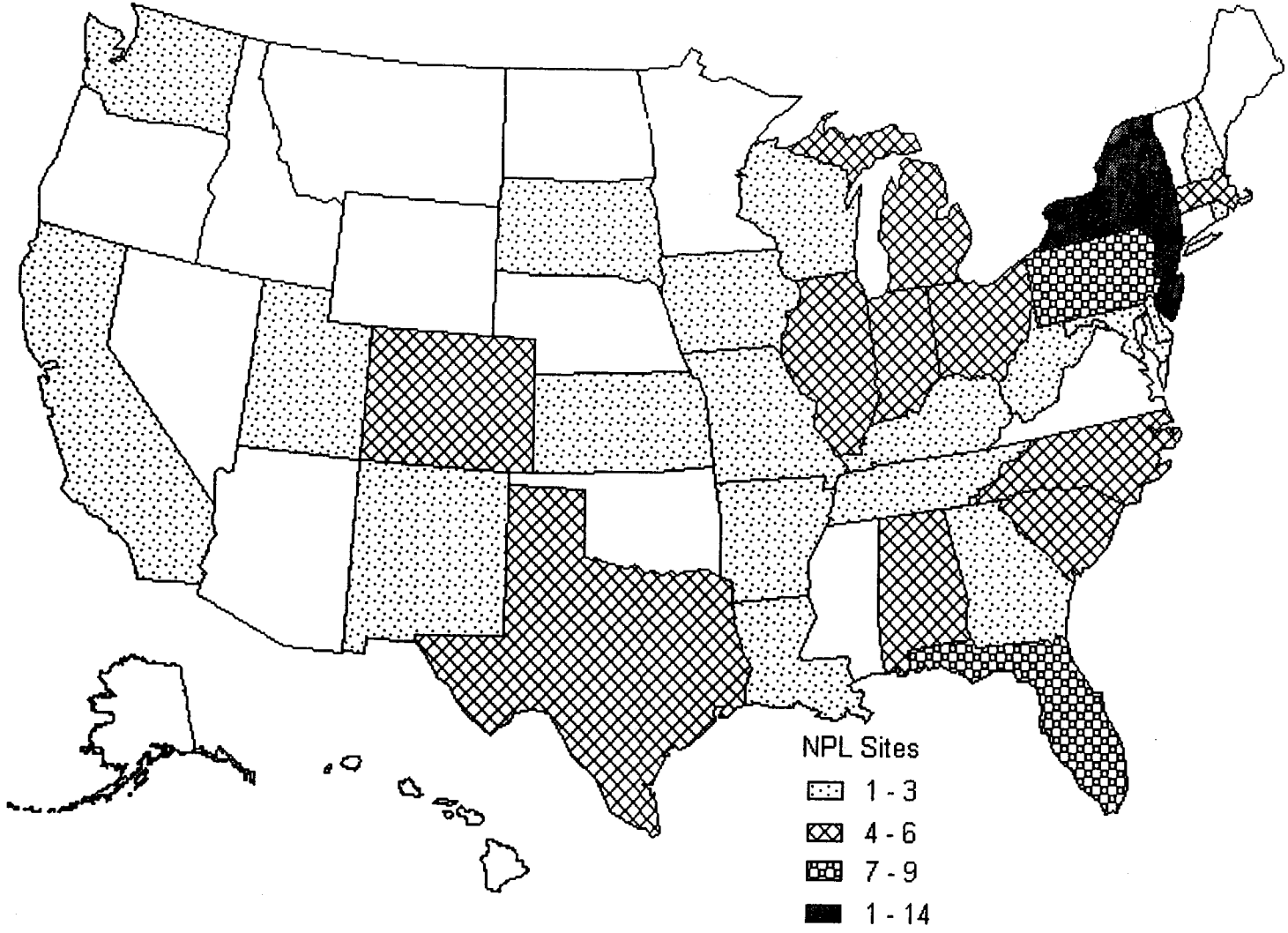
Lindane and other isomers of HCH do not occur naturally in the environment. Most current releases of lindane in the United States are related to its formulation and its use as an insecticide/acaricide.

Figure 5-1. Frequency of NPL Sites with Gamma-HCH Contamination



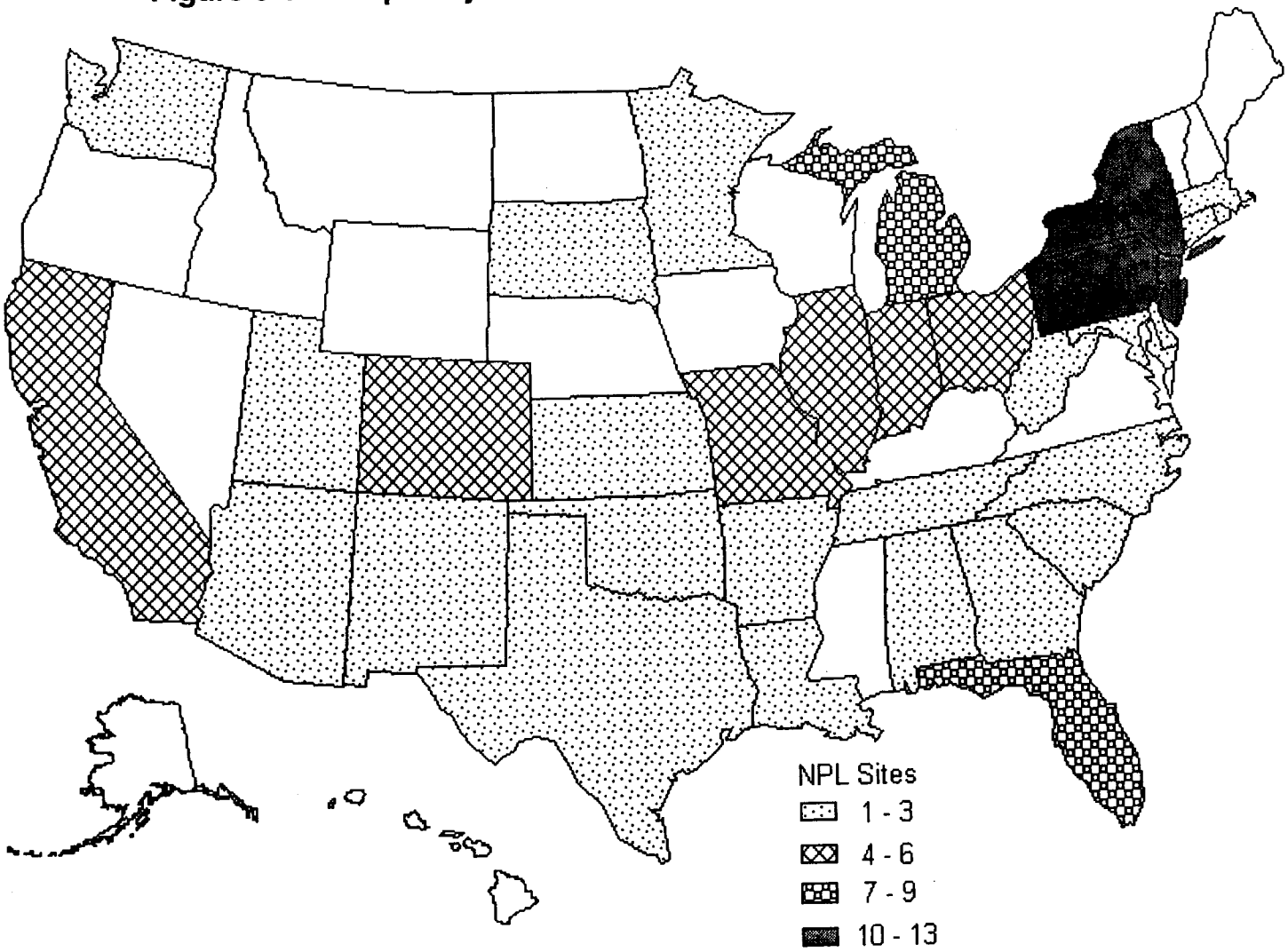
*Derived from HAZDAT 1998

Figure 5-2. Frequency of NPL Sites with Alpha-HCH Contamination



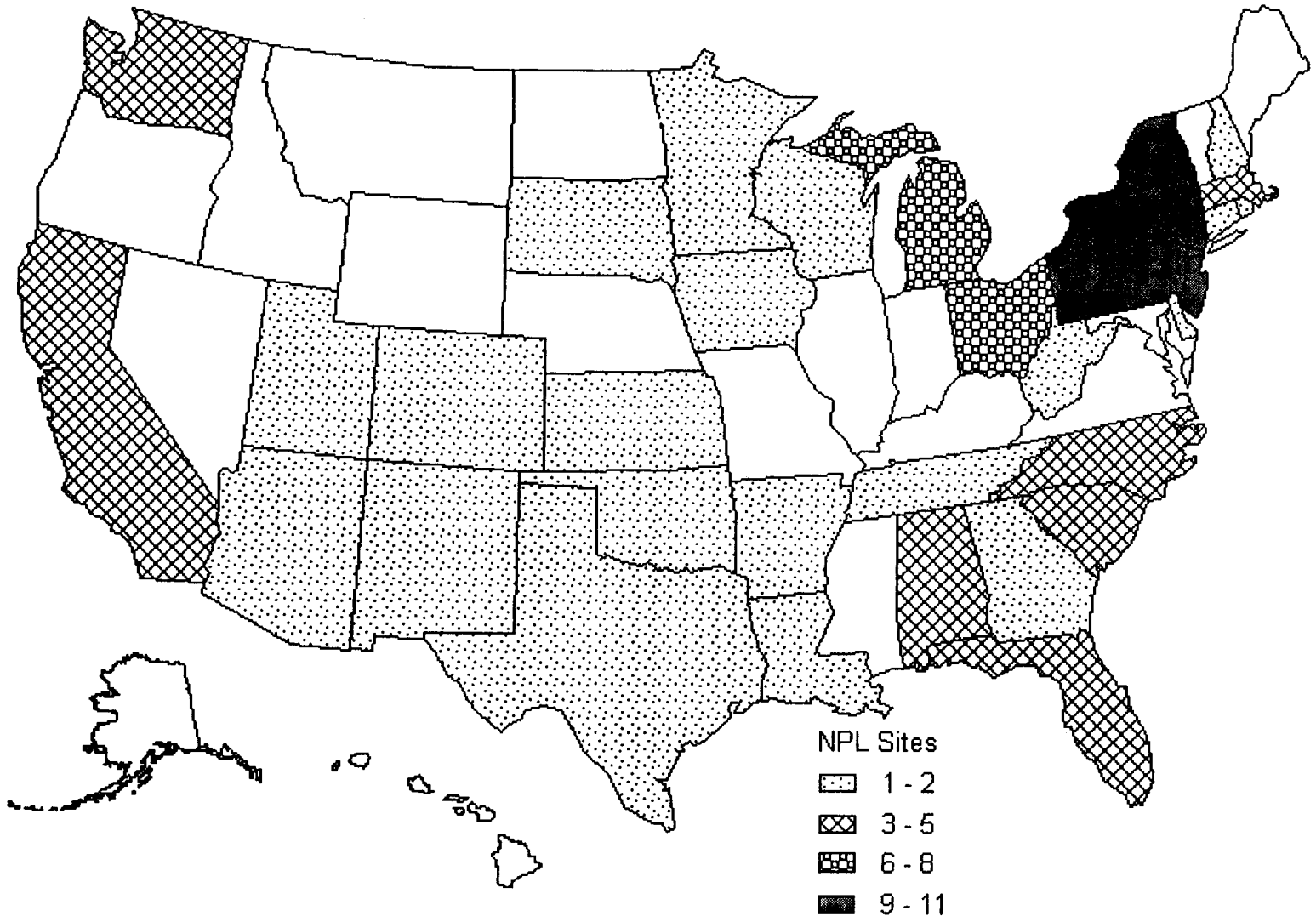
* Derived from HAZDAT 1998

Figure 5-3. Frequency of NPL Sites with Beta-HCH Contamination



*Derived from HAZDAT 1998

Figure 5-4. Frequency of NPL Sites with Delta-HCH Contamination



*Derived from HAZDAT 1998

Table 5-1. Releases to the Environment from Facilities that Manufacture or Process Hexachlorocyclohexane

			Total of reported amounts in pounds per year[a]						
State[b]	City	Facility	Air[c]	Water	Land	Underground Injection	POTW Transfer	Off-Site Waste Transfer	Total Environment[d]
GA	Cordele	Drexel Chemical Co.	10	5	250	0	0	0	265
ID	Marsing	Gustafson Inc.	0	0	0	0	0	414	414
KY	Buckner	Rigo Co.	0	0	0	0	0	250	250
NE	Fremont	Platte Chemical Co.	500	0	0	0	0	1,000	1,500
		TOTALS	510	5	250	0	0	1,664	2,429

Source: TRI96 1998

[a]Data in TRI are maximum amounts released by each facility
[b]Post office state abbreviations used
[c]The sum of fugitive and stack releases are included in releases to air by a given facility
[d]The sum of all releases of the chemical to air, land, and water, and underground injection well; and transfers off-site by a given facility

POTW = publicly owned treatment works

5.2.1 Air

According to the Toxic Chemical Release Inventory, in 1996, releases of lindane to the air from four large processing facilities were 510 kg (1,133 pounds) (TRI96 1998). Table 5-1 lists amounts released from these facilities. The TRI data should be used with caution because only certain types of facilities are required to report. This is not an exhaustive list.

Historically, the largest source of γ-HCH releases to the air resulted from agricultural application of the pesticide lindane. Other air releases occurred during the manufacture of the pesticide. Aerial applications of γ-HCH are now prohibited in the United States as its use as a pesticide was restricted (EPA 1985b), and atmospheric releases from these sources are not expected. α-HCH and γ-HCH were detected in 60–90% of the air samples collected in the vicinity of formulation plants in Arkansas and Tennessee in 1971 at mean levels of 1.0 and 1.3 mg/m^3, respectively (Lewis and Lee 1976). Quantitative estimates of the total quantities of γ-HCH released to the air from these sources were not located.

In addition to releases from industrial facilities, γ-HCH is present in the environment as a result of its use or disposal. For example, wind erosion of contaminated soil may distribute pesticides into the atmosphere. γ-HCH can also be released to the atmosphere via volatilization from treated agricultural soils and plant foliage (Lewis and Lee 1976). Evaporative loss of γ-HCH from water is not considered a significant source of atmospheric γ-HCH because of its relatively high water solubility (Mackay and Leinonen 1975). Quantitative estimates of the amount of γ-HCH released from these sources were not located in the literature. Atmospheric release of γ-HCH from disposal sites or hazardous waste sites has not been documented but is likely, considering the physical and chemical properties of γ-HCH.

α, β, γ, and δ-HCH have been detected in air samples collected at 5, 3, 6, and 3 of the 1,467 current or former EPA NPL hazardous waste sites, respectively (HazDat 1998).

5.2.2 Water

According to the Toxic Chemical Release Inventory, in 1996, releases of lindane to the water from four large processing facilities were 5 kg (11 pounds) (TRI96 1998). Table 5-1 lists amounts released from these

facilities. The TRI data should be used with caution because only certain types of facilities are required to report. This is not an exhaustive list.

γ-HCH can be released to surface water via surface runoff (as the dissolved chemical or adsorbed to particulates) or via wet deposition of rain and snow (Tanabe et al. 1982; Wheatly and Hardman 1965). For example, Lake Ontario received 7 kg/year of α-HCH and <2 kg/year of γ-HCH because of suspended sediment loading from the Niagara River between 1979 and 1981 (Kuntz and Warry 1983). The Great Lakes in general receive from 0.77 to 3.3 metric tons/year of α-HCH and from 3.7 to 15.9 metric tons/year of γ-HCH because of atmospheric deposition of these contaminants (Eisenreich et al. 1981). In 1982, α-HCH and γ-HCH were detected in samples of urban stormwater runoff from Denver, Colorado, and Washington, DC, at 0.0027–0.1 and 0.052–0.1 µg/L in 20% and 11%, respectively, of the 86 samples collected; β-HCH was detected in runoff from Washington, DC, only, in 5% of the samples at a concentration of 0.1 µg/L (Cole et al. 1984).

γ-HCH can be released to groundwater via soil leachate. Although available adsorption data indicate that γ-HCH has a low mobility in soils, the results of monitoring studies suggest that γ-HCH does migrate to groundwater (Page 1981; Sandhu et al. 1978) (see Section 5.4.2). In water tested from 1,076 wells throughout New Jersey, γ-HCH was not detected in at least half of the samples, but a maximum concentration of 0.9 ppb γ-HCH was detected (Page 1981).

α, β, γ, and δ-HCH have been detected in groundwater samples collected at 61, 60, 77, and 58 of the 1,467 current or former EPA NPL sites, respectively (HazDat 1998). α, β, γ, and δ-HCH have been detected in surface water samples collected at 26, 16, 30, and 10 of the 1,467 current or former EPA NPL sites, respectively (HazDat 1998).

5.2.3 Soil

According to the Toxic Chemical Release Inventory, in 1996, releases of lindane to the soil from eight large processing facilities were 250 kg (556 pounds) (TRI96 1998). Table 5-1 lists amounts released from these facilities. The TRI data should be used with caution because only certain types of facilities are required to report. This is not an exhaustive list.

γ-HCH can be released to the soil by direct application of the pesticide to soil or by direct or indirect releases during formulation, storage, and/or disposal. Hazardous waste sites where γ-HCH has been disposed of in the past are sources of γ-HCH in soils (HAZDAT 1992). However, the application of lindane (purity unspecified) to laboratory refuse columns simulating municipal landfills indicated that lindane did not volatilize or leach from the refuse surface, and movement through the column was slight, suggesting that codisposal of lindane with municipal refuse will result in minimal releases (Reinhart and Pohland 1991; Reinhart et al. 1991).

α, β, γ, and δ-HCH have been detected in sediment samples collected at 13, 16, 30, and 20 of the 1,467 current or former EPA NPL sites, respectively (HazDat 1998). α, β, γ, and δ-HCH have been detected in soil samples collected at 51, 64, 77, and 50 of the 1,467 current or former EPA NPL sites, respectively (HazDat 1998). Also, α, β, γ, and δ-HCH have been detected in leachate collected at 7, 8, 12, and 9 of the 1,467 current or former EPA NPL sites, respectively (HazDat 1998).

5.3 ENVIRONMENTAL FATE

5.3.1 Transport and Partitioning

γ-HCH present in soil can leach to groundwater, sorb to soil particulates, or volatilize to the atmosphere. In general, the leaching of organic chemicals through soil is governed by the water solubility of the chemicals and their propensity to bind to soil. Based on the results of a number of laboratory soil column leaching studies that used soils of both high and low organic carbon content as well as municipal refuse, γ-HCH is generally immobile in soils (Hollifield 1979; Melancon et al. 1986; Rao and Davidson 1982; Reinhart et al. 1991). Adsorption of γ-HCH to soil particulates is generally a more important partitioning process than leaching to groundwater. However, groundwater sediments, which have low organic carbon content, are not sufficient to adsorb γ-HCH to the extent that groundwater contamination is prevented (Nordmeyer et al. 1992). In a study involving a laboratory sediment/water system, α- and γ-HCH isomers were highly adsorbed on sediments under both aerobic and anaerobic conditions (Wu et al. 1997).

γ-HCH sorbed to the soil can partition to the atmosphere by wind erosion of surface soil particulates (Stanley et al. 1971) and via volatilization from treated agricultural soils and plant foliage (Lewis and Lee 1976). In tests conducted in a model laboratory system at 10EC and 20EC, volatilization half-lives of γ-HCH from soil and oat plant surfaces of 2.3–24.8 days and 0.29–0.73 days, respectively, were reported (Dorfler et al. 1991a);

half-lives were greater on dry, sandy soils versus peat soils; however, when moisture was added to the soils, the half-life was greater for the peat soil, while the warmer temperature decreased the half-life under all soil and moisture conditions (Dorfler et al. 1991b). In tests performed with a wind tunnel, a volatilization rate of >20% for lindane from soil surfaces within a 24-hour period was determined (Rüdel 1997). The volatilization rate from plant surfaces was 55% for lindane. Application of γ-HCH to fields of sunflowers and sugarbeets resulted in a 54% evaporative loss of the pesticide within 24 hours (Neururer and Womastek 1991).

An analysis of the concentrations of α-HCH to γ-HCH in air over southern Ontario suggested that high levels of γ-HCH were indicative of recent lindane usage (Hoff et al. 1992a). The levels of α-HCH were less variable throughout the year, ranging from 77–260 pg/m^3. During the winter, higher ratios of α-HCH to γ-HCH reflect the movement of air containing the more persistent α-HCH isomer from the colder Arctic regions to the south, while the lower ratios in the summer reflect both increased lindane usage in the region and the lack of movement of Arctic air (Hoff et al. 1992a). γ-HCH is also seen to move with warm air during the summer months from the lower United States (or areas even further to the south) to the Great Lakes region, although a similar trajectory cannot be identified for the more ubiquitous α-HCH. Levels of α-HCH in air are not dominated by volatilization or partitioning to surfaces but are dependent on local temperature changes (Hoff et al. 1992b). α-HCH appears to have a long residence time in the atmosphere and is controlled primarily by transport.

γ-HCH in the atmosphere is likely to be subject to rain-out and dry deposition. γ-HCH removal rates by rainfall and dry deposition were 2.5%/week and 3.3%/week, respectively, and the estimated residence time of γ-HCH in the atmosphere was 17 weeks in a study by Atkins and Eggleton (1971). Rain-out and dry deposition of atmospheric γ-HCH results in the contamination of surface soil and water in areas not directly exposed via pesticide application. γ-HCH concentrations were positively correlated with ambient air temperature although concentrations of α-HCH were not.

In surface waters, γ-HCH has a tendency to dissolve and remain in the water column. Although γ-HCH has a relatively high vapor pressure compared with many other organochlorine insecticides, evaporative loss of γ-HCH from water is not considered to be significant. Mackay and Leinonen (1975) calculated theoretical losses of several pesticides from saturated water solutions and predicted a volatilization half-life of 191 days for γ-HCH.

γ-HCH released to water may undergo adsorption/desorption with sediments and other materials in the water. Adsorption and desorption studies of γ-HCH in natural water-sediment systems performed by Saleh et al. (1982) indicate that a diversity of the natural water-sediment characteristics may affect the sorption-desorption behavior of γ-HCH in addition to the organic carbon content of the sediments. Lindane is sorbed to silt solutions with a slow desorption rate, indicating that transport through the environment is most likely to be particle mediated (Noegrohati and Hammers 1992c). Biosorption of lindane was seen for the fungus *Rhizopus arrhizus* and activated sludge, with equilibrium being reached within 1 and 4 hours, respectively. Death of the sludge biomass resulted in rapid desorption with zero-order kinetics, suggesting that adsorbed lindane can be released back into the environment (Tsezos and Wang 1991a). The sorption of lindane from water using wood charcoal has been described (Keerthinarayana and Bandyopadhyay 1998); it was found to be a good sorbent for the sorption of lindane from water.

Lindane which is adsorbed to sediments may be recycled to the atmosphere as gas bubbles are formed in the sediment by the methanogenesis and denitrification processes of bacteria. It is estimated that in one case studied 85% of the lindane associated with the sediment gas bubbles will be released to the atmosphere, with the remaining 15% being dissolved in the water column as the bubble rises toward the surface (Fendinger et al. 1992).

γ-HCH is bioconcentrated to high levels following uptake from surface waters by a number of aquatic organisms. However, uptake from soils and bioconcentration by plants and terrestrial organisms appears to be limited. For example, bioconcentration factors (BCFs) for γ-HCH from surface waters include 183 in brine shrimp (Matsumura and Benezet 1973), 319 in rainbow trout fry (Ramamoorthy 1985), 84 in pink shrimp, 218 in pinfish, 63 in grass shrimp, and 490 in sheepshead minnows (Schimmel et al. 1977). Introduction of γ-HCH onto sand resulted in a BCF of 95 in brine shrimp and 1,613 in northern brook silverside fish (Matsumura and Benezet 1973). A BCF of 1,273 (lipid basis) in prawns (crustacean) was seen to be 0.58 times the γ-HCH concentration in the underlying sediment, indicating that although aquatic organisms may accumulate γ-HCH from the water column, uptake from contaminated sediment alone may not be extensive (Just et al. 1990). BCFs for the isomers of HCH, using zebra-fish under steady-state conditions, were 1,100 for α-HCH, 1,460 for β-HCH, 850 for γ-HCH, and 1,770 for δ-HCH; BCFs determined by uptake and clearance rate constants were slightly lower (Butte et al. 1991). BCFs on a wet weight basis for γ-HCH in different fish species were positively correlated with their lipid content (Geyer et al. 1997). The bioaccumulation of lindane by tubificide oligochaetes from a static system consisting of sediment and water has been reported (Egeler et al. 1997).

γ-HCH applied to an aquatic mesocosm (i.e., a small, artificial ecosystem) at 61.3 µg/L was reduced by 50% at 24 hours postapplication, while at 19 weeks postapplication the concentration in the water was only 0.2%, and no γ-HCH was detected at 21 weeks. The biological half-life was estimated to be 16.7 days. Movement through the water column was shown by increasing sediment concentrations up to a maximum of 75.4 µg/kg at 96 hours postapplication; however, sediment concentrations decreased to below the detection limit at 23 weeks to give a half-life in sediment of 48.1 days. Rooted aquatic macrophytes have a BCF of 56 at a maximum concentration of 1.7 mg/kg at 24 hours postapplication; however, at 14 weeks all residues were below the detection limit for a half-disappearance time of 18 days. Gastropods in the system had a maximum γ-HCH concentration of 7.2 mg/kg at 24 hours posttreatment, yielding a BCF of 232.4 and a half-disappearance time of 13.7 days with all residues eliminated by 13 weeks (Caquet et al. 1992).

In tests with radiolabeled γ-HCH, grain, maize, and rice plants accumulated 0.95%, 0.11%, and 0.04%, respectively, of the amount of bound residues following 14–20 days growth in a sandy loam soil. Bioconcentration increased by 4–10 times when the plants were grown in test soils containing both bound and extractable residues of γ-HCH (Verma and Pilli 1991). Plants and grains grown on soil treated with γ-HCH showed α-HCH as the predominant isomer although all isomers were found to some extent; amounts decreased with increasing time after application (Singh et al. 1991).

Uptake of γ-HCH by earthworms from a treated humus soil has also been reported. Following exposure to 5 ppm of the compound for up to 8 weeks, the test organisms bioconcentrated γ-HCH by a factor of 2.5. The earthworms biotransformed more than 50% of the accumulated γ-HCH; the main degradation product was γ-2,3,4,5,6-pentachlorocyclohex-1-ene (Viswanathan et al. 1988).

γ-HCH and the other isomers of HCH do not appear to undergo biomagnification in terrestrial food chains to a great extent, although there is a moderate potential for transfer of γ-HCH to animal tissue as a result of soil ingestion or ingestion of contaminated foliage (Wild and Jones 1992). Clark et al. (1974) found that γ-HCH levels in the adipose tissue of cattle were 10 times higher than in the feed (0.002 mg/kg). Szokolay et al. (1977) examined relative accumulation of HCH isomers including γ-HCH and various components in the food chain in Czechoslovakia. Lower γ-HCH residues were found in tissues of animals (chickens, sheep, pigeons) feeding entirely on plant material whereas carnivores had higher concentrations.

The effect of soil loading (the amount of soil deposited per unit area of skin) on the dermal bioavailability of γ-HCH from contaminated soils has been examined (Duff and Kissel 1996). A static *in vitro* diffusion

apparatus and abdominal skin from human cadavers were used. Results indicated that the dermal absorption of γ-HCH from soil is dependent on soil loading and was estimated to be 0.45–2.35%. Dermal absorption of γ-HCH increased significantly with decreases in soil loading providing monolayer or greater coverage of the skin is maintained.

5.3.2 Transformation and Degradation

5.3.2.1 Air

As mentioned earlier, γ-HCH can be present in the air as vapor or sorbed to particulate matter. The widespread global distribution of HCH isomers is indicative of the persistence of γ-HCH in the air. It appears that photodegradation or other degradation processes are not significant in the removal of γ-HCH from air, as compared to rain-out or dry deposition. However, Hamada et al. (1981) found that γ-HCH underwent photodegradation to form two isomers of tetrachlorohexene and pentachlorohexene in propanol solution when irradiated with ultraviolet light produced by a low-pressure mercury lamp. Similar transformation of γ-HCH and other isomers may occur, to some extent, in the atmosphere.

5.3.2.2 Water

Biodegradation is believed to be the dominant degradative process for γ-HCH in aquatic systems, although hydrolysis and photolysis do occur. Sharom et al. (1980) found that <30% of the applied γ-HCH remained in unsterilized natural waters in capped bottles after 16 weeks. Biodegradation was concluded to be responsible for these results, although it was unclear to what extent hydrolysis or adsorption to the glass bottles may have contributed to the results. Zoetemann et al. (1980) estimated river, lake, and groundwater half-lives for γ-HCH from degradation data in these environments to be 3–30 days, 30–300 days, and >300 days, respectively. In natural lake water with a pH of 9.0 and a hardness of greater than 600 mg calcium carbonate/liter, the half-life of γ-HCH was estimated to be 65 hours (Ferrando et al. 1992). Lindane, applied at concentrations of 50 or 500 µg/L to aerobic batch cultures of microorganisms with sodium acetate as a carbon source, was initially removed by adsorption and followed by desorption onto the biomass with subsequent decomposition (McTernan and Pereira 1991). Approximately 56–62% of the lindane was removed from the water column in 23 days, with 26% removal by adsorption onto the biological solids produced in these batch reactors. Microbial growth, using γ-HCH in the absence of sodium acetate, increased as the microorganisms

became acclimated, the pesticide still showed toxic properties, as evidenced by a concurrent increase in microbial death rates.

It has been shown that γ-HCH is degraded by nitrogen-fixing blue-green algae. These algae reduce the toxic effects of γ-HCH following repeated inoculations (Kar and Singh 1979b). The degradation of γ-HCH became more efficient with time, thus reducing the pesticide's toxicity in cultures of nitrogen-fixing blue-green algae. Dechlorination of γ-HCH to γ-pentachlorocyclo-hexene was also shown to occur with fungi in aqueous suspensions (Machholz and Kujawa 1985) and in algal cultures (Sweeney 1969).

Hydrolysis is not considered an important degradation process for γ-HCH in aquatic environments under neutral pH conditions. However, under alkaline conditions, γ-HCH is hydrolyzed fairly rapidly. Saleh et al. (1982) tested rates of hydrolysis of γ-HCH in sterilized natural waters at 25EC and found that hydrolysis of γ-HCH followed first-order kinetics with half-lives of 92 hours at pH 9.3, 648 hours at pH 7.8, and 771 hours at pH 7.3.

Somewhat conflicting information is available on the rate of photolysis of γ-HCH in water. In the study by Saleh et al. (1982) discussed above, the authors also reported γ-HCH first-order photolysis half-lives of 169, 1,791, and 1,540 hours at pH 9.3, 7.3, and 7.8, respectively. The adjusted midwinter half-life of γ-HCH in pure water was reported to be 1,560 hours. However, in another study, γ-HCH rapidly disappeared from a sterile aqueous solution when exposed to ultraviolet radiation in atmospheric nitrogen; less than 1% of the original amount was left in solution after 30 hours of exposure (Malaiyandi et al. 1982). Photolysis of lindane in aqueous solution in the presence of polyoxomethallate and ultraviolet light has been demonstrated (Hiskia et al. 1997).

5.3.2.3 Sediment and Soil

γ-HCH in soil or sediment is degraded primarily by biotransformation; however, the major removal mechanism for γ-HCH from soils, at least in warm climates, is the volatilization of the compound from soil surfaces. A 6-fold increase in γ-HCH volatilization from soil was seen when the temperature increased from 15EC to 45EC; flooding the soil also increased the volatilization (Samuel and Pillai 1990). Tu (1976) reported that 71 of 147 microorganisms isolated from a loamy sand soil were able to utilize a γ-HCH solution as the sole carbon source. White rot fungus degraded radiolabeled γ-HCH in aerobic pure culture laboratory tests. In a silt loam soil/corncob test matrix, 34.7% of the compound was degraded over a 60-day

test period, whereas 53.5% degradation was observed in liquid cultures over a 30-day test period (Kennedy et al. 1990). The results of this study have been confirmed by more recent studies (Mougin et al. 1996; Mougin et al. 1997). The isolation of γ-HCH-degrading bacteria, classified as *Sphingomonas paucimobilis*, from contaminated soils has been reported (Thomas et al.1996). A *Pseudomonas* species has also been isolated from pretreated soil that is able to degrade γ-HCH and α-HCH, but not β-HCH, within 10–20 days under both flooded (anaerobic) and unflooded (aerobic) conditions; greater degradation rates were observed under aerobic conditions (Sahu et al. 1993). However, the concentrations and persistence of γ-HCH in soil are dependent on soil types. An analysis of two soil types, loamy sand (approximately 1–2% organic matter) and muck (approximately 27–56% organic matter), for γ-HCH residues showed that mean residues in the loamy sand soil had decreased from 95 ppb dry weight in 1971 to below the detection limit of 10 ppb in 1989; however, in muck, residues had decreased from 426 ppb in 1971 to 168 ppb in 1989 (Szeto and Price 1991). The presence of crops on the soils also affects the persistence of HCH residues, with half-lives of 58.8 days and 83.8 days for cropped and uncropped plots, respectively. β-HCH was the most persistent isomer with half-lives of 184 and 100 days, respectively, on cropped and uncropped plots; γ-HCH was next at 107 and 62.1 days, followed by α-HCH at 54.4 days and 56.1 days, and finally, δ-HCH at 33.9 and 23.4 days. Only trace amounts of the isomers were found to leach below 20 cm soil depth (Singh et al. 1991). The β-HCH isomer comprised 80–100% of the total HCH residues found in soil or vegetation on land surrounding an industrial landfill in Germany 10 years after the final HCH input (Heinisch et al. 1993).

Most available information suggests that γ-HCH transformation is favored in biologically rich, anaerobic environments (Callahan et al. 1979; Haider 1979; Kalsch et al. 1998). In bench-scale anaerobic digestion tests designed to assess the fate of semivolatile organic pollutants in primary and secondary sludges, γ-HCH was found to undergo 98% degradation at 120 days. Sorption of the compound to the digester solids accounted for 2% of the initial feed; none of the compound was lost by volatilization. The digesters were operated at 35EC with a 30-day solids retention time (Govind et al. 1991). Similar results were seen with live activated sludge where initially reversible biosorption dominates the removal process followed by an increased aerobic biodegradation after approximately 10 hours of acclimation. The biodegradation process includes hydrolytic dechlorination with subsequent ring cleavage and finally, partial or total mineralization (Tsezos and Wang 1991b). Adaptation of sewage sludge is slow and may take 1–2 months; however, once acclimation occurs, 70–80% biodegradation of γ-HCH may occur, with the percentage of degradation decreasing with increasing sludge age (Nyholm et al. 1992). Co-oxidation or reductive dechlorination are the probable degradation mechanisms (Jacobsen et al. 1991; Nyholm et al. 1992).

Numerous diverse studies on biological degradation have shown that γ-HCH was transformed to tetrachlorohexene; tri-, tetra-, and pentachlorinated benzenes; penta- and tetra cyclohexanes; other isomers of HCH; and other related chemicals. The products varied depending on what organisms were present, what products were sought, and when the sample was analyzed (Callahan et al. 1979). Laboratory studies have demonstrated the bioisomerization of γ-HCH to α-, β-, and δ-HCH but bioisomerization in the environment was considered to be nonsignificant by an investigator who conducted a field study (Waliszewski 1993). Levels of individual isomers were approximately 0.1–1.4% and 0.8–4.0% of the γ-HCH concentrations at 3–31 weeks and 34–46 weeks, respectively, following γ-HCH treatment of soil. An inability to control all environmental conditions in the laboratory was discussed as a possible reason for differences in results between laboratory and field studies.

Abiotic transformation and degradation processes of γ-HCH in soil/sediment are not thought to be significant pathways. As discussed earlier for water, photolysis or hydrolysis are not considered important degradation pathways of γ-HCH and other isomers.

5.4 LEVELS MONITORED OR ESTIMATED IN THE ENVIRONMENT

Reliable evaluation of the potential for human exposure to hexachlorocyclohexane depends in part on the reliability of supporting analytical data from environmental samples and biological specimens. In reviewing data on hexachlorocyclohexane levels monitored or estimated in the environment, it should also be noted that the amount of chemical identified analytically is not necessarily equivalent to the amount that is bioavailable.

5.4.1 Air

γ-HCH was detected in ground level ambient air samples collected in College Station, Texas, in 1979–1980 at a mean concentration of 0.23 ng/m^3 (range, 0.01–1.60 ng/m^3) (Atlas and Giam 1988). The compound has also been detected in troposphere air samples collected over the Adirondack Mountains in New York state in 1985 at a mean concentration of 0.509 ng/m^3 and over Newport News, Virginia, in 1988 at a mean concentration of 0.021 ng/m^3 (Knap and Binkley 1991). Air monitoring over southern Ontario, Canada, from July 1988 to July 1989 showed annual mean air concentrations of α-, β-, and γ-isomers to be 0.145, 0.0018, and 0.06 ng/m^3 with a total HCH annual mean concentration of 0.21 ng/m^3 and with the greatest total HCH concentrations during the summer months (Hoff et al. 1992a).

In a study of global distribution and atmospheric transport of chlorinated hydrocarbons in the West Pacific, Eastern Indian, and Antarctic Oceans, Tanabe et al. (1982) confirmed the widespread distribution of HCH isomers. HCH residues were detected in all 79 air and water samples collected. The concentrations ranged from 1.1 to 2.0 ng/m^3 in air and from 3.1 to 7.3 ng/L in water. Other monitoring studies include the detection of γ-HCH in the lower troposphere over the Southern Indian Ocean in 1986 at a mean concentration of 0.406 ng/m^3 (Wittinger and Ballschmiter 1990), in the lower troposphere over Bermuda in 1988 at a mean concentration of 0.012 ng/m^3 (Knap and Binkley 1991), and in ambient air samples collected at Axel Hieberg Island in the Canadian arctic at 0.017–0.07 ng/m^3 (Hargrave et al. 1988).

γ-HCH has also been detected in rainfall samples collected in College Station, Texas, in 1979–1980 at a weighted mean concentration of 2.81 ng/L (range, 0.30–7.8 ng/L) (Atlas and Giam 1988) and in Bermuda in 1983–1984 at a mean concentration of 0.126 ng/L (range, 0.001–0.936 ng/L) (Knap et al. 1988). In rainfall samples collected at four sites in Canada in 1984, γ-HCH concentrations ranged from 0.46 to 34 ng/L (Strachan 1988). The mean concentration in rainfall samples collected at Lake Superior during the 1984 wetfall season was 3.0 ng/L, with an annual loading of 2.0 μg/m^2/year (Strachan 1988). These values were less than those determined in the years 1977, 1981, and 1983 (Strachan 1988). γ-HCH has been detected in rain and snow water in Portland, Oregon in 1982 at mean concentrations ranging from 0.45 to 11 ng/L (Pankow et al. 1984). Rainwater collected in Hawaii in 1970–1971 had a mean γ-HCH concentration of 5 ng/L, with concentrations ranging from 1 to 19 ng/L (Bevenue et al. 1972). Snow and ice samples collected at Axel Hiberg Island in the Canadian Arctic in 1986 contained γ-HCH at concentrations of 0.211–0.644 ng/L and 0.186 ng/L, respectively (Hargrave et al. 1988). Rain samples collected in Germany between June 1990 and August 1991 contained γ-HCH at a mean concentration of 0.208 μg/L (range, 0.020–0.833 μg/L; detection limit, 0.5 pg) in 39 of 41 samples (Scharf et al. 1992).

5.4.2 Water

Surface water concentrations of γ-HCH have been measured in many areas across the United States. Reported concentrations ranged from 10 to 319 parts per ton (ppt) (mean concentration of 147 ppt) in Hampton County, South Carolina (Sandhu et al. 1978), to much higher concentrations of 0.052–0.1 parts per billion (ppb) in Washington, DC, and Denver (Cole et al. 1984). The majority of the available monitoring studies were conducted in the early to mid 1970s. The most recent monitoring study was conducted in 1980–1981 in the Niagara River near its entry into Lake Ontario. In that study, γ-HCH was detected in 99% of all samples at a mean concentration of 2.1 ppt (Kuntz and Warry 1983). γ-HCH concentration in Lake

Michigan tributary streams ranged from undetected to 0.15 ppb (Schacht et al. 1974). According to EPA's STORET database, γ-HCH was detected in 27% of 4,505 surface water samples collected in the United States at a median concentration of 0.020 µg/L (Staples et al. 1985). γ-HCH concentrations in groundwater samples were greatest in the West South Central region (Phillips and Birchard 1991). The compound was also found in water samples collected in Lake Ontario in 1983 at 0.806–1.85 ng/L concentration (Biberhofer and Stevens 1987).

γ-HCH has been detected in more than 10% of urban stormwater runoff samples in two U.S. cities at concentrations between 0.052 and 0.1 ppt (Cole et al. 1984). In urban runoff samples collected in the Canadian Great Lakes Basin, γ-HCH was detected at mean concentrations of 0.0065 µg/L and 0.0035 mg/kg in the aqueous and sediment portions, respectively; the mean annual loading of the compound in runoff in the basin was reported to be 4.1 kg/year (Marsalek and Schroeter 1988).

γ-HCH has been detected in groundwater at a median concentration of 16 ppt in Chesterfield County, South Carolina, and 163 ppt in Hampton, South Carolina (Sandhu et al. 1978). A concentration range of undetected to 0.9 ppt was reported for groundwater samples from New Jersey. γ-HCH has also been detected in drinking water from Cincinnati, Ohio (Keith et al. 1976); Hampton, South Carolina (Sandhu et al. 1978); and Oahu, Hawaii (Bevenue et al. 1972), at mean concentrations of 0.01 ppt, 10 ppt, and 0.2 ppt, respectively. In a study of α-HCH and γ-HCH in Saskatchewan, Canada, these HCH isomers were not detected frequently in surface waters that originate from ground water (Donald et al. 1997).

5.4.3 Sediment and Soil

According to EPA's STORET database, γ-HCH was detected in 0.5% of 596 sediment samples collected throughout the United States at a median concentration of <2.0 µg/kg (Staples et al. 1985). According to data collected in STORET between 1978 and 1987, γ-HCH was found in the greatest concentration in sediment from the West North Central census region of the United States, followed by the Mountain region and the East South Central region (Phillips and Birchard 1991). γ-HCH was detected in 33% of suspended sediment samples collected from the Niagara River; the average concentration was 2 ppb (Kuntz and Warry 1983). The average γ-HCH concentration in settling particulates from Lake Ontario was 2.4 ppb in 1982 (Oliver and Charlton 1984). Sediment samples from Lake St. Francis on the St. Lawrence River contained a mean total HCH concentration of 0.6 ng/g dry weight (range, <0.1–2.0 ng/g), suggesting that deposition of contaminated materials from Lake Ontario was of less importance than local inputs of HCH (Sloterdijk

1991). γ-HCH concentrations in creek sediments collected in 1976 near the James River in Virginia ranged from 7.3 to 8.5 ppb (Saleh et al. 1978). γ-HCH was included in the analytes monitored in the National Oceanic and Atmospheric Administration's (NOAA) Status and Trends Mussel Watch Program conducted in the Gulf of Mexico. The compound was detected in 19% of the sediment samples collected in 1987 at a mean concentration of 0.07 ng/g (median, <0.02 ng/g; range, <0.02–1.74 ng/g) (Sericano et al 1990). Sediment samples collected around the Great Lakes in May 1989, contained γ-HCH concentrations ranging from below the detection limit (0.10 µg/kg) to 0.99 µg/kg (wet weight) (Verbrugge et al. 1991). Thirty-three sediment samples from 11 impoundments along the Indian River Lagoon in Florida contained γ-HCH at concentrations ranging from 34.4 ng/g in the top layer of sediment at one impoundment to 9.4 ng/g in the bottom layer at the same site (Wang et al. 1992). The pesticide lindane had been used for mosquito control in the area from the late 1950s to the mid 1960s. Interstitial water samples from the impoundment sites did not contain detectable levels of the pesticide.

5.4.4 Other Environmental Media

γ-HCH residues were detected in fat samples of domestic farm animals collected in Ontario, Canada, in 1986–1988. Mean concentrations in fat from chickens, turkeys, beef, lamb, and pork ranged from 0.012 to 0.032 mg/kg; the mean concentration in hen eggs was 0.008 mg/kg (Frank et al. 1990b).

Residues of γ-HCH on tomatoes decreased by 23.9%, 15 days after application of the pesticide (from 195.6 µg/kg to 148.8 µg/kg). Processing the tomatoes (e.g., pureeing, making tomato juice) reduced the residue levels by 100% after the waiting period; however, washing the tomatoes reduced the residues by up to 55.9% (Bessar et al. 1991). A pesticide residue screening program carried out by the H.E.B. Food Stores of San Antonio between 1989 and 1991 detected γ-HCH in 4 of 429 onion samples (detection limit, 0.02 ppm); however, none of the positive samples exceeded the action level for this commodity (Schattenberg and Hsu 1992).

As part of NOAA's Status and Trends Mussel Watch Program conducted in the Gulf of Mexico, γ-HCH was detected in 80% of the oyster samples collected in 1987 at a mean concentration of 1.74 ng/g (median, 1.20 ng/g; range, <0.25–9.06 ng/g) (Sericano et al. 1990). Samples taken in 1992 from Mexico's Palizada River, located in a major agricultural area with substantial pesticide use, contained an average γ-HCH concentration of 0.08 ng/g in shrimp but no detectable levels in oysters or mussels (Gold-Bouchot et al. 1995). Combined concentrations of other HCH isomers were found to be 1.18 ng/g in shrimp, 1.04–1.97 ng/g in oysters, and

1.68 ng/g in mussels. Schmitt et al. (1985) reported the results of a monitoring study of fish tissues from 107 freshwater stations in the United States. A decline in tissue occurrence of detectable α- and γ-HCH residues was observed from 1976 to 1981. During 1980–1981, whole body residues of γ-HCH exceeded 0.01 ppb at only one station, where levels were 0.02–0.03 ppb. Tissue concentrations of α-HCH were higher than γ-HCH. The highest concentrations for α-HCH were 0.03–0.04 ppb and were found in fish from the southwestern and midwestern United States. An analysis of fish from the Upper Steele Bayou in Mississippi in 1988 indicated that β-HCH concentrations ranged from undetected to 0.02 mg/kg wet weight in fish; no β-HCH was detected in snakes or sediments taken from the same area (Ford and Hill 1991). Atlantic cod taken from relatively isolated stock in the southern Gulf of St. Lawrence showed declining tissue concentrations of α-HCH between 1977 (1.865 µg/kg) and 1985 (1.792 µg/kg).

An analysis of pesticide residues in green coffee and after roasting indicated that technical-grade HCH was found in green coffee at concentrations ranging from <0.005 ppm to 0.204 ppm. However, storage and roasting reduced the pesticide residues by 60–67% and up to 98%, respectively, with darker roasting resulting in the greatest reduction (McCarthy et al. 1992).

5.5 GENERAL POPULATION AND OCCUPATIONAL EXPOSURE

Human exposures to γ-HCH can result from the ingestion of plants, animals, animal products, milk, and water containing the pesticide. Farm animals may be exposed to the compound through feed, air, or water or cutaneous application for protection from ectoparasites. Lipophilic pesticides such as γ-HCH accumulate in adipose tissue. Clark et al. (1974) found that γ-HCH levels in the adipose tissue of cattle were 10 times higher than in the feed (0.002 mg/kg). An analysis of data from 238 families in Missouri between June 1989 and March 1990, indicated that 9.2% of the families reported using Kwell shampoo (contains γ-HCH) for lice control on children (Davis et al. 1992).

The most likely route of nonmedicinal human exposure to γ-HCH is ingestion of food containing the pesticide. A smaller degree of exposure may result from ingestion of drinking water containing γ-HCH. For example, γ-HCH was detected in 6% of the foods collected in eight market basket surveys from different regions of the United States during the period of April 1982 to April 1984 (Gunderson 1988). Foods representative of eight infant and adult population groups were prepared for consumption prior to analysis in a revision to FDA's Total Diet Studies methodology. The estimated mean daily intakes (ng/kg body weight/day) of γ-HCH for these groups in 1982–1984 were as follows: (1) 6–11-month-old infants, 1.9;

(2) 2-year-old toddlers, 7.9; (3) 14–16-year-old females, 3.1; (4) 14–16-year-old males, 3.4; (5) 25–30-year-old females, 2.0; (6) 25–30-year-old males, 2.5; (7) 60–65-year-old females, 1.6; and (8) 60–65-year-old males, 1.8. γ-HCH intakes (ng/kg body weight/day) for three of these groups in 1988 were estimated in the FDA's Total Diet Analyses to be as follows: (1) 6–11-month-old infants, 0.8; (2) 14–16-year-old males, 1.4; and (3) 60–65-year-old females, 0.9 (FDA 1989b). HCH isomers have been detected in the following feed types formulated for infants and toddlers: whole milk and other dairy products; meat, fish, and poultry; oils and fats; vegetables; and sugars and adjuncts (Gartrell et al. 1986a).

HCH isomers were also detected in adult diet foodstuffs, including dairy products; meat, fish, and poultry; garden fruits; oils and fats; leafy and root vegetables; and sugar and adjuncts (Gartrell et al. 1986b). Daily intake values of HCH isomers in adult diets in 1981–1982 were reported to be 0.010 μg/kg/day for total HCH; 0.008 μg/kg/day for α-HCH; <0.001 μg/kg/day for β-HCH and δ-HCH; and 0.002 μg/kg/day for γ-HCH. In the Total Diet Study conducted by FDA in 1990 on 936 food items, γ-HCH was detected in 23 items, while α-HCH and β-HCH (combined) were detected in 11 items. Information on the amount of levels found were not provided (Yess 1991). The average concentration of lindane in 234 ready-to-eat foods was 0.0012 μg/g (KAN-DO Office and Pesticides Team 1995).

Studies in which soils containing 10 ppm radiolabeled γ-HCH were added to human skin samples at a quantity that exceeded complete coverage (5 mg soil / cm^2 skin) demonstrated mean γ-HCH absorptions of 1.04% from sandy soils and 1.64% from silt soils (Duff and Kissel 1996). However, data from soil absorption studies can vary due to factors such as the amount of soil added to skin, the exposure time, and possible evaporation of the contaminant.

The results of biomonitoring studies can be used as indicators of human exposures to HCH. The National Human Adipose Tissue Survey (NHATS) conducted in 1982 showed that β-HCH (the most prevalent HCH isomer in fatty tissue) was detected in 87% of 46 composite samples at <19–570 ng/g (ppb) concentrations (Stanley 1986). It was detected most often in postmortem samples collected from individuals from the southern United States. In another survey conducted in 1970–1975, β-HCH was detected in more than 90% of the postmortem human adipose tissue samples at an average level of 300 ppb (Kutz et al. 1979). In a review of the NHATS data available from 1970 to 1983, Mack and Mohadjer (1985) reported that the estimated 1983 national median level of β-HCH was 80 ppb, in comparison to the historic level of 140 ppb. The median level has decreased over time, but the compound has continued to be detected in nearly 100% of the population surveyed. Median levels are highest in the South census region and tend to increase with age

but have not been found to differ across the sexes or racial groups. A further analysis of the NHATS data indicated that average β-HCH concentrations in fat had decreased from 0.45 ppm in 1970 to approximately 0.16 ppm since 1981 (Kutz et al. 1991).

A comparison of the levels of α-HCH and β-HCH in the whole blood and biopsy fat of 25 patients showed median levels of 0.04 ng/g (maximum, <0.04 ng/g) and 0.13 ng/g (maximum, 2.60 ng/g) for the blood and 1.1 ng/g (maximum, 9.6 ng/g) and 18.0 ng/g (maximum, 748.6 ng/g) for the fat tissue, respectively (Mes 1992). A further comparison of β-HCH levels in breastmilk and adipose tissue samples was made for populations living near the Great Lakes (Canada only) and in other Canadian regions. Mean β-HCH levels in breast milk (0.6 ng/g) and adipose tissue (23.4 ng/g) were lower near the Great Lakes than in other parts of Canada (0.8 ng/g and 30.8 ng/g, respectively) (Mes and Malcolm 1992). Levels of HCHs in the adipose tissue of Japanese males increased from the late 1940s to 1966, coinciding with an increased annual production of HCH (Loganathan et al. 1993). Levels have been dropping since HCHs were banned in 1971, from a maximum level of 28 μg/g to present levels of less than 1 μg/g. Since 1974, only the more persistent β-HCH isomer has been found (Loganathan et al. 1993).

γ-HCH was one of the most frequently detected pesticides in the blood of Virginia residents, although the number of individuals sampled was not identified (Griffith and Blanke 1975). γ-HCH blood concentrations were the highest in residents of the middle age group (41–60 years). Some of the frequency of γ-HCH occurrence in the state was attributed to its common use in commercial vaporizers and its presence in cigarette smoke (Griffith and Blanke 1975). The National Health and Nutrition Examination Survey (NHANES) analyzed blood and urine specimens for the presence of HCH isomers. β-HCH was detected in approximately 13.9% of the U.S. population (12–74 years) in the Northeast, Midwest, and South. The median level for the 91% quantifiable positive results was 1.7 ppb (Murphy and Harvey 1985).

Factors such as age, dietary habits, and residence can influence the body burden of γ-HCH in exposed individuals. In one study, it was shown that women between the ages of 26 and 34 years who lived in a rural area of India and were nonvegetarians tended to show higher body levels of γ-HCH than other Indian women who lived in an urban area or who were vegetarians (Saxena et al. 1981a). The higher levels of γ-HCH in women at an older child-bearing age suggest that a longer life span may cause a greater accumulation of pesticide in the body. Higher pesticide levels are found in mutton, eggs, and chicken which are common in nonvegetarian meals; therefore, there tends to be a higher level of γ-HCH in the bodies of nonvegetarians. Individuals living in rural areas are more likely to be exposed to γ-HCH because agricultural fields are the

primary site of application of pesticides. In addition, studies indicate that γ-HCH is also present in breastmilk at an average level of 0.006 ppm in Alberta, Canada (Currie et al. 1979). In a study of 50 donors of breastmilk in Oahu, Hawaii, Takahashi et al. (1981) demonstrated HCH in 82% of the samples at a mean level of 81 ppb within a range of 0–480 ppb, expressed in terms of extractable lipid.

A study conducted in Colorado indicated, in general, that no quantitative relationships were demonstrated between pesticide levels in household dust and pesticide levels in blood. However, γ-HCH levels in blood sera in a pesticide formulator (16.8 ppb) and his wife (5 ppb) were found to be elevated in a household in which dust levels measured 5.85 ppb (Starr et al. 1974). It is possible that the γ-HCH found in the wife's blood and in the household came from the clothes and person of the pesticide formulator.

The Nonoccupational Pesticide Exposure Study (NOPES) conducted by EPA was based on the Total Exposure Assessment Methodology (TEAM) approach to exposure estimation. NOPES was designed to provide estimates of nonoccupational exposure to 32 household pesticides in the United States. Samples were collected at two locations: (1) Jacksonville, Florida, an area representative of high pesticide usage; and (2) Springfield/Chicopee, Massachusetts, an area of low-to-moderate pesticide usage. Detectable levels of γ-HCH were found in the personal air samples of 32–70% of the Jacksonville sample population; the range of mean concentrations in the air samples was 7–22 ng/m^3. For the Springfield population, detectable levels of γ-HCH were found in personal air samples collected from 8% to 10% of the population, with mean concentrations of 0.7–5 ng/m^3 (EPA 1990c).

A study on occupational pesticide exposure of commercial seed-treating applicators was conducted in Montana (Grey et al. 1983). No exposure was detectable on the chest and arm pads, but γ-HCH was detected on the hands and on the respirator pads. Workers involved with γ-HCH application complained of nasal irritation if they did not wear a respirator or mask. The α-, β-, γ-, and δ-isomers of HCH have been detected in the blood serum and adipose tissue of individuals occupationally exposed to HCH in pesticide formulation. Serum levels of <0.5 ppb–1 ppm α-HCH, <0.9 ppb–0.72 ppm β-HCH, <0.7 ppb–0.17 ppm γ-HCH, and 0.002–0.16 ppm δ-HCH have been detected in exposed workers (Baumann et al. 1980; Kashyap 1986; Morgan and Lin 1978; Nigam et al. 1986). Mean adipose tissue levels of 5.8 mg α-HCH/kg, 45.6 mg β-HCH/kg, and 3.1 mg γ-HCH/kg have also been reported in exposed workers (Baumann et al. 1980).

In general, accidental or intentional ingestion would lead to the highest exposures. Worker exposure constitutes the next highest exposure population although worker exposure is decreasing in both the number

of workers exposed and the levels of exposure. Lastly, the general population receives the lowest levels, which occur mainly from ingestion of foods and water with γ-HCH residues. Living near a waste disposal site contaminated with γ-HCH will also increase the likelihood of exposure.

5.6 EXPOSURES OF CHILDREN

This section focuses on exposures from conception to maturity at 18 years in humans and briefly considers potential pre-conception exposure to germ cells. Differences from adults in susceptibility to hazardous substances are discussed in 2.6 Children's Susceptibility.

Children are not small adults. A child's exposure may differ from an adult's exposure in many ways. Children drink more fluids, eat more food, and breathe more air per kilogram of body weight, and have a larger skin surface in proportion to their body volume. A child's diet often differs from that of adults. The developing human's source of nutrition changes with age: from placental nourishment to breast milk or formula to the diet of older children who eat more of certain types of foods than adults. A child's behavior and lifestyle also influence exposure. Children crawl on the floor, they put things in their mouths, they may ingest inappropriate things such as dirt or paint chips, they spend more time outdoors. Children also are closer to the ground, and they do not have the judgement of adults in avoiding hazards (NRC 1993).

Prenatal exposure of children to HCH can occur. β- HCH and γ-HCH have been found in samples of human maternal adipose tissue, maternal blood, cord blood, and breastmilk in women who were exposed to unknown levels of various organochlorine pesticides in Kenya (Kanja et al. 1992). Placental transfer of HCH in humans has been well documented (Saxena et al. 1981). Higher levels of total HCH and lindane were found in specimens of maternal blood, placenta, and umbilical-cord blood from women experiencing premature labor, spontaneous abortions, and stillbirths when compared to matched controls (Saxena et al. 1980; Saxena and Siddiqui 1983). Saxena et al. (1980) reported HCH levels of 69.3–550.4 ppb and γ-HCH levels of 30.8–113.6 ppb in the blood of women in India who had experienced spontaneous abortions or premature labor compared with blood HCH levels of 22.2–85.5 ppb and γ-HCH levels of 7.1–32.5 ppb in women who had undergone full-term pregnancy. Serum levels of a number of other pesticides including aldrin, DDE, DDT, and DDD were also found to be higher in cases of premature labor and spontaneous abortions. It was, therefore, not possible to establish a causal relationship between the serum HCH levels and these adverse effects. However, HCH has been shown to accumulate in amniotic fluid, placenta, and fetal tissues after treatment of pregnant mice (Srivastava and Raizada 1993) and can be related to fetolethality.

HCH is commonly detected in low concentrations (0.015 mg/kg fat) in the breastmilk of women exposed to HCH in the environment (Fytianos et al. 1985). Levels of HCH isomers in breastmilk have been reported, particularly in developing countries that still use HCH as a pesticide. Studies indicate the γ-HCH is present in breastmilk at an average level of 6 ppb in Alberta, Canada (Currie et al. 1979). In a study of 50 donors of breastmilk in Oahu, Hawaii, Takahashi et al. (1981) demonstrated HCH in 82% of the samples at a mean level of 81 ppb within a range of 0–480 ppb, expressed in terms of extractable lipids. Breastmilk concentrations of α-, β -, γ-, and δ-HCH were determined from samples obtained from two areas of India that were under malaria control (Dua et al. 1997). The mean concentrations of α-, γ-, β-, and δ-HCH in one area were 0.002, 0.002, 0.022, and 0.001 (mg/kg) , while in the second area concentrations were 0.003, 0.006, 0.078, and 0.002, respectively. Another study performed in a different region of India also demonstrated the presence of HCH isomers in breastmilk (Nair et al. 1996). Mean breastmilk concentrations of α-, β-, and γ-HCH were 0.045, 0.198 and 0.084 (mg/L), respectively. δ-HCH was not detected in the breastmilk samples. In a study designed to quantify the levels of organochlorine residues in the breastmilk of mothers in Uganda, Africa, the milk fat concentrations of α- HCH, β-HCH and γ-HCH ranged from 0.006–0.46, 0.005–0.25 and 0.01–0.87 mg/kg, respectively (Ejobi et al. 1996). The concentration of β-HCH in breastmilk samples from 3 regions in the Czech Republic ranged from 71 to 80 ng/g (Schoular et al. 1996). A comparison of β-HCH levels in breastmilk and adipose tissue samples was made for populations living near the Great Lakes (Canada only) and the rest of Canada. Mean β-HCH levels in breastmilk (0.6 ng/g) and adipose tissue (23.4 ng/g) were lower near the Great Lakes than in other parts of Canada (0.8 ng/g and 30.8 ng/g, respectively) (Mes and Malcolm 1992).

As mentioned previously, exposures to HCH can result from the ingestion of plants, animals, animal products, milk, and water containing the pesticide. A smaller degree of exposure may result from ingestion of drinking water containing HCH. There is also the possibility of exposure to γ-HCH from medical usage (e.g., shampoos for control of lice and lotion for treatment of scabies). Numerous studies have documented the effects in humans overexposed to γ-HCH through misuse or accidental ingestion of products used to treat head lice (Davies et al. 1983; Jaeger et al. 1984; Lee and Groth 1977). Although some controversy exists as to whether γ-HCH is a safe therapeutic agent when used in accordance with the manufacturers' guidelines, it is clear that most exposures occur through misuse of products (Rasmussen 1980, 1981, 1987). Besides medical usage, children are likely to be exposed to HCH from the ingestion of food containing the pesticide. Based on FDA's Total Diet Analyses, γ-HCH intakes (body weight/day) are 0.8 µg/kg for 6-11-month-old infants, 7.9 µg/kg for 2-year-old toddlers, and 1.4 and 3.1 µg/kg for 14–16-year-old males and females, respectively (FDA 1989b). HCH isomers have been detected in the following food types formulated for

infants and toddlers: whole milk and other dairy products; meat, fish, and poultry; oils and fats; vegetables; and sugars and adjuncts (Gartrell et al. 1986a).

HCH isomers have also been detected in cow's milk in those countries that still use the chemical as a pesticide. In a study performed in Uganda, Africa, the concentrations of α- HCH, β-HCH and lindane in cow's milk were 0.002–0.014, 0.003–0.018, and 0.006–0.036 mg/kg milkfat, respectively (Ejobi et al. 1996). Mean levels of HCH isomers analyzed in cow's milk samples from 2 separate areas in India were 0.0045 and 0.012 mg/kg α-HCH, 0.002 and 0.015 mg/kg γ-HCH, 0.0105 and 0.028 mg/kg β-HCH and 0.002 and 0.003 mg/kg δ-HCH (Dua et al. 1997). A monitoring study of 192 samples of cow's milk from Mexico revealed 0.001–0.201 mg/kg α-HCH, 0.008–0.253 mg/kg β-HCH and 0.002–0.187 mg/kg γ-HCH (Waliszewski et al. 1996). HCH isomers have also been detected in buttermilk and butter prepared from cow's milk contaminated with these isomers (Sreenivas et al. 1983).

HCH is bioavailable from soil and can be absorbed both orally and dermally (Duff and Kissel 1996). γ-HCH exhibited mean 24-hour dermal absorption values from 0.45 to 2.35% varying with different soil types and soil loadings of 1, 5, and 10 mg/cm^3. Some children intentionally eat dirt and most inadvertently ingest dirt by putting fingers or other objects in their mouths while playing outdoors. Thus, they are more likely than adults to be exposed to HCH via ingestion or direct contact of soil contaminated with HCH.

Children may also be exposed to a significant amount of HCH from household dust; parents' work clothes, skin, hair, tools, and other objects removed from the workplace are a likely source of exposure to children. An analysis of environmental contribution to pesticide body burden indicated household dust can be a major source of environmental HCH exposure (Starr et al. 1974), as indicated by elevated γ-HCH levels in blood sera in a pesticide formulator (16.8 ppb) and his wife (5 ppb) in a household in which dust levels measured 5.85 ppb. It is possible that the γ-HCH found in the wife's blood and in the household came from the clothes and person of the pesticide formulator.

γ-HCH is a restricted use pesticide. Its registered use around the home is limited to structural treatment, dog shampoo, and dog dust for fleas and ticks. Children can be exposed at home because of its potential use on pets and improper or illegal pesticide application.

Analyses of blood samples of 186 children living in an area contaminated with HCH, which was used as an insecticide in Brazil, revealed the presence of α-, γ-, and β-, HCH isomers (Brilhante and Oliveira 1996).

The authors reported that 24% of the children showed 0.89 ppb average concentrations of β-HCH in the blood. α- and γ-isomers were detected in only 3 and 1 children, respectively, at a mean concentration of 1.8 ppb and 0.95 ppb, respectively.

5.7 POPULATIONS WITH POTENTIALLY HIGH EXPOSURES

The populations with the most potential for chronic exposure to HCH are workers who either manufacture or routinely use these isomers. Exposure of the general population to γ-HCH tends to be low because federal regulations limiting its use have taken effect. However, γ-HCH is available in some consumer products (e.g., shampoos, food) and medications, and the possibility of exposure from these products is a source of concern. Individuals living near hazardous waste sites contaminated with γ-HCH may also be exposed to the compound.

Numerous studies have documented the effects in humans overexposed to γ-HCH through misuse or accidental ingestion of products used to treat scabies and head lice (Davies et al. 1983; Jaeger et al. 1984; Lee and Groth 1977). Although some controversy exists as to whether γ-HCH is a safe therapeutic agent when used in accordance with the manufacturers' guidelines, it is clear that most exposures occur through misuse of products (Rasmussen 1980, 1981, 1987). In addition, other studies have described cases in which patients have shown neurotoxic effects following excess exposure or ingestion of pesticides (Harris et al. 1969; Hayes 1976; West 1967).

Exposure to the other isomers of HCH (as in the technical-grade HCH) is limited in the United States as a result of regulations restricting their use. However, persons traveling or living in areas where the use of HCH is legal (e.g., South America, Eastern Europe, and Asia) should be wary of exposure to isomers of HCH through food and drinking water sources (Krauthacker et al. 1986; Radomski et al. 1971a; Saxena et al. 1980, 1981a, 1981b).

5.8 ADEQUACY OF THE DATABASE

Section 104(i)(5) of CERCLA, as amended, directs the Administrator of ATSDR (in consultation with the Administrator of EPA and agencies and programs of the Public Health Service) to assess whether adequate information on the health effects of hexachlorocyclohexane is available. Where adequate information is not available, ATSDR, in conjunction with the NTP, is required to assure the initiation of a program of research

designed to determine the health effects (and techniques for developing methods to determine such health effects) of hexachlorocyclohexane.

The following categories of possible data needs have been identified by a joint team of scientists from ATSDR, NTP, and EPA. They are defined as substance-specific informational needs that if met would reduce the uncertainties of human health assessment. This definition should not be interpreted to mean that all data needs discussed in this section must be filled. In the future, the identified data needs will be evaluated and prioritized, and a substance-specific research agenda will be proposed.

5.8.1 Identification of Data Needs

Physical and Chemical Properties. Sufficient information is available on the physical and chemical properties of γ-HCH and the other HCH isomers (see Chapter 3) to permit an assessment of the environmental fate of these compounds. No additional studies are required at this time.

Production, Import/Export, Use, Release, and Disposal. Production methods for HCH are well described in the literature (IARC 1979). γ-HCH is used as an insecticide and as a therapeutic scabicide and pediculicide for treatment of ectoparasite in humans and animals (Budvari et al. 1989). The production and use of γ-HCH as a pesticide has been restricted in the United States, and the use of technical-grade HCH was voluntarily canceled in 1976 (EPA 1978). There is no current information on the import of γ-HCH, and there is no information on the import of other HCH isomers. This information will be helpful for estimating human exposure particularly of populations living near industrial and hazardous waste sites. Release of γ-HCH to environmental media has been primarily from its use as a pesticide. Wastes containing γ-HCH must be contained, incinerated, and disposed of in landfills (EPA 1991g). Carbon absorption or flocculation are useful treatment methods for the removal of HCH from aqueous effluent streams, except when methanol is also contained in the effluents (HSDB 1993). Disposal methods are currently subject to revision under EPA guidance.

According to the Emergency Planning and Community Right-to-Know Act of 1986, 42 U.S.C. Section 11023, industries are required to submit chemical release and off-site transfer information to the EPA. The Toxics Release Inventory (TRI), which contains this information for 1994, became available in

May of 1996. This database will be updated yearly and should provide a list of industrial production facilities and emissions.

Environmental Fate. γ-HCH released to the environment partitions to the atmosphere, soils, and sediments (Atkins and Eggleton 1971; Lewis and Lee 1976; Melancon et al. 1986; Saleh et al. 1982; Stanley et al. 1971). The compound is transported in the atmosphere, surface water, and groundwater (Mackay and Leinonen 1975; Nordmeyer et al. 1992; Stanley et al. 1971). γ-HCH is transformed via biodegradation in soils and surface waters (Govind et al. 1991; Kar and Singh 1979b; Kennedy et al. 1990; Macholz and Kujawa 1985; Sharom et al. 1980; Tu 1976). Available data indicate that photodegradation or other degradation processes are not significant processes in the removal of γ-HCH from air, as compared to rain-out and dry deposition (Atkins and Eggleton 1971; Hamada et al. 1981). Additional information on the transport, transformation, and persistence of the compound in soils and groundwater, particularly at hazardous waste sites, would be useful in identifying the most important routes of human exposure to γ-HCH. There is information regarding the half-lives for γ-HCH in water (3–30 days, 30–300 days, and >300 days for river, lake, and groundwater, respectively [Zoetemann et al. 1980]), but not in air or soil. There is no information about the half-lives for the other HCH isomers in any environmental media. Environmental fate data on HCH isomers other than γ-HCH are scant. Additional data on the half-lives for γ-HCH in air and soil, and further environmental fate data for the other HCH isomers, would be helpful. These data could be used to estimate exposure to HCH under various conditions of environmental release for purposes of planning and conducting meaningful follow-up exposure and health studies.

Bioavailability from Environmental Media. Evidence of absorption following inhalation and dermal exposure is available for workers involved in the formulation of pesticide products containing HCH isomers and in the use of γ-HCH (Baumann et al. 1980; Grey et al. 1983). Dietary intake is a major route of exposure for the general population (Gunderson 1988). Additional information on the absorption of γ-HCH, following ingestion of foods containing residues of the compound, would be helpful. As mentioned in Section 5.3.1, Duff and Kissel (1996) showed that bioavailability of γ-HCH via dermal exposure depended upon levels of soil loading. Dermal absorption ranged from 0.45 to 2.35%. For populations living in the vicinity of hazardous waste sites, additional information on absorption following dermal contact with, or ingestion of, contaminated soil would also be helpful, given the expected strong sorption of the compound to soil particulates. Besides γ-HCH, other isomers of HCH have been detected in adult diet foodstuffs (Gartrell et al. 1986b). Additional information on the absorption of these other HCH isomers following ingestion of foods containing residues of these isomers would be helpful. Because of the potential of HCH to contaminate

air, drinking water, and soil, further information on the bioavailability of the HCH isomers from these environmental media would be useful for assessing possible health concerns for humans.

Food Chain Bioaccumulation. γ-HCH in surface waters and soils is taken up and bioconcentrated by terrestrial and aquatic organisms (Just et al. 1990; Matsumura and Benezet 1973; Ramamoorthy 1985; Verma and Pillai 1991; Viswanathan et al. 1988). γ-HCH is bioconcentrated to high levels following uptake from surface waters by a number of aquatic organisms (Matsumura and Benezet 1973; Ramamoorthy 1985; Schimmel et al. 1977). Uptake from soils and bioconcentration by plants and terrestrial organisms appears to be limited (Verma and Pillai 1991; Wild and Jones 1992). Limited information suggests that the compound is not biomagnified in terrestrial food chains because of its metabolism by terrestrial organisms (Schmitt et al. 1985). Bioconcentration values in zebra-fish for α-HCH and β-HCH are reported (Butte et al. 1991). Among the HCH isomers, β-HCH accumulates the most in the food chain (Szokolay 1977). Additional information on the potential bioaccumulation of α-, β-, and δ-HCH isomers in terrestrial and aquatic food chains would be helpful.

Exposure Levels in Environmental Media. Environmental monitoring data are available predominantly for γ-HCH in air (Atlas and Giam 1988; Knap and Binkley 1991), surface water (Sandhu et al. 1978; Staples et al. 1985), groundwater (Sandhu et al. 1978), soil (Carey et al. 1978; Staples et al. 1985), and foods (FDA 1989b; Gunderson 1988; Kutz et al. 1976). γ-HCH has been detected in air, surface water and groundwater, and sediment and soil. The widespread distribution of HCH isomers in air has been confirmed (Tanabe et al. 1982). Although the use of γ-HCH has been restricted and the use of technical-grade HCH was voluntarily canceled in 1976 (EPA 1978), it is not likely that new environmental measurements will show considerably lower levels of γ-HCH in these media since there are remaining impacts from importing and processing HCH. Therefore, additional information on the levels of γ-HCH and α-, β-, and δ-HCH isomers is needed to assess the current potential human exposure to the chemicals from environmental media, particularly near hazardous waste sites.

Reliable monitoring data for the levels of hexachlorocyclohexane in contaminated media at hazardous waste sites are needed so that the information obtained on levels of hexachlorocyclohexane in the environment can be used in combination with the known body burdens of hexachlorocyclohexane to assess the potential risk of adverse health effects in populations living in the vicinity of hazardous waste sites.

Exposure Levels in Humans. HCH can be detected in the blood (Baumann et al. 1980; Griffith and Blanke 1975; Murphy and Harvey 1985), urine (Murphy and Harvey 1985), adipose tissue (Baumann et al. 1980; Stanley 1986), breastmilk (Takahasi et al. 1981), and semen (Stachel et al. 1989) of exposed individuals. Most of the data on the body burden of HCH are from adipose tissue and blood serum analyses conducted postmortem or on occupationally exposed individuals. The disadvantage of using postmortem blood is that the HCH concentration may change after death. The occupational studies often do not report environmental levels; therefore, it is not possible to correlate body HCH levels with environmental levels. The results of the National Human Adipose Tissue Survey (NHATS) conducted in 1982 showed that β-HCH, the most prevalent isomer in fatty tissue, was detected most often in postmortem samples collected from individuals from the southern U.S. Additional information is needed on exposure to γ-HCH and α-, β-, and δ-HCH isomers in populations living in the vicinity of hazardous waste sites.

This information is necessary for assessing the need to conduct health studies on these populations.

Exposures of Children. The different pathways for exposure of children to HCH have been discussed in Section 5.6. Prenatal exposure of children to HCH has been demonstrated; it is well documented that placental transfer of HCH occurs, and HCH levels have been measured in placenta and cord blood in humans (Saxena et al. 1981; Nair 1996) and in amniotic fluid and fetal tissues in mice (Srivastava and Raijada 1993). Infants may also be exposed via ingestion of breastmilk and cow's milk. Exposure may also occur via ingestion of water containing HCH, food and animal products, and possibly through incidental ingestion of household dust. It has been demonstrated that household dust can be an important source of environmental HCH (Starr et al. 1974). This occurs especially if the parents work in facilities that process or use HCH and can bring home residues of HCH via their work clothes, skin, hair, tools, or other objects removed from the workplace. A take-home exposure study on pesticide applicators might be useful if such occupational exposure settings occur. Limited studies conducted on exposure of infants and children to γ-HCH from application of 1% γ-HCH lotion as scabicide indicated dermal absorption occurred (Ginsberg et al. 1977). Adipose tissue is a major storage depot for HCH. Although data from a national human adipose tissue survey exist (Stanley 1984), no quantitative data are currently available on the body burden of HCH in children. These studies are needed because unique exposure pathways for children exist, and children may be different from adults in their weight-adjusted intake of HCH because of their higher surface area to volume ratio and higher ingestion rate of household dust.

Exposure Registries. No exposure registries for hexachlorocyclohexane were located. This substance is not currently one of the compounds for which a subregistry has been established in the National Exposure Registry. The substance will be considered in the future when chemical selection is made for subregistries to be established. The information that is amassed in the National Exposure Registry facilitates the epidemiological research needed to assess adverse health outcomes that may be related to exposure to this substance.

5.8.2 Ongoing Studies

As part of the Third National Health and Nutrition Evaluation Survey (NHANES III), the Environmental Health Laboratory Sciences Division of the National Center for Environmental Health, Centers for Disease Control and Prevention, will be analyzing human blood samples for hexachlorocyclohexane and other volatile organic compounds. These data will give an indication of the frequency of occurrence and background levels of these compounds in the general population.

Biodegradability of HCH isomers and engineering applicability are being investigated in the Netherlands. Also, remedial investigations and feasibility studies at the NPL sites known to have γ-HCH contamination should add to the available database for environmental levels, environmental fate, and human exposure.

Ongoing studies concerning the environmental fate of HCH isomers have been identified as follows:

C.A. Reddy (Michigan State University) is currently examining a lignin-degrading filamentous fungus *(Phanerochaete chrysosporium)* to isolate, characterize, and develop expression/secretion systems for ligninases. These ligninases play a key role in lignin degradation and are also believed to be involved in the detoxification of γ-HCH. Similar studies on the biodegradation of γ-HCH by the white rot fungus are also being conducted by S.D. Aust at Utah State University.

W.F. Spencer (Agricultural Research Service, Riverside, California) is conducting studies in the laboratory and field to quantify the physical, chemical, and biological parameters related to rates of volatilization, degradation, and transport of γ-HCH and other chemicals into the atmosphere.

The effects of γ-HCH and 14 other insecticides on transformations of urea nitrogen (urea hydrolysis and nitrification) in 2 coarse-textured and 2 fine-textured soils are currently being examined by J.M. Brenner (Iowa State University).

M. Speedie and B. Pogell (University of Maryland) are investigating the metabolism of γ-HCH by streptomycetes. They have found degradative detoxification of 80% of γ-HCH added within 5 days; 60% is degraded by the end of the first 24 hours. The investigators assume that degradation occurs via a lignin peroxidase-mediated reaction.

R.W. Coble of the U.S. Geological Survey is conducting a study of the hydrology of an area containing six pesticide disposal sites near Aberdeen, Maryland. γ-HCH has been identified in water samples collected from several municipal wells in the area.